principles for its therapeutics to rest on. It is perhaps this spirit which allows the disastrous use of palliatives, iodine, alcohol, and, as one writer recommends,[16] common vinegar.

It is a fact that cauterization of any kind, that is, the application of anything that has the essential quality of caustics, is a valuable means of saving life. According to the writer in Eulenburg's *Real-Encyclopadie,* many are saved in this way in Germany; according to Hogyes cauterization has increased the percentage of cures from 19 to 41 per cent.; according to Proust,[17] "Of 117 people bitten but not cauterized 76 died; while of 249 cauterized only 89 died," all of which shows the high empirical probability of the value of cauterization.

Statistics, it is obvious, do not finally solve problems. Instances gathered in this way make simple calculations of probability not causal connections. It is a method auxiliary to scientific induction so-called, and a causal connection between cauterization and a certain uniformity of result may be inferred by comparing the averages of cases in animals with the average of especially enumerated cases in humans, which are conditioned by the same factor. This process leads us to the possibility of establishing strict uniformity among cases cauterized and cases protected from hydrophobia.

THE MEDICAL ASPECT OF CHRONIC TYPHOID INFECTION (TYPHOID BACILLUS CARRIERS).[1]

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IT has proved interesting and instructive in connection with the subject of this paper to review the opinions held by teachers of medicine twenty-five years ago relative to certain infectious diseases. Until about two decades ago studies in the problems of immunity were hardly begun, while the science of bacteriology was in its infancy. In fact, because of the rapid development of the science rather than the art of medicine, it may be safely asserted that more progress has been made in our understanding of the early and late effects of infectious diseases during this period than had been possible during the centuries preceding. This progress has been made possible through the labors of the early investigators, and it is more fortunate for the coming generations than we can conceive that there existed such creative minds as those of Koch, Pasteur, Virchow, Ehrlich, and Wright.

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[16] Töpfer, Klin. Jahrb., 1907.

[17] Annales de l'Institut Pasteur, 1894, 435.

[1] Read before the Mississippi Valley Medical Association at Nashville, Tennessee, October 17, 1911.

We now understand, tentatively at least, many facts not available even a decade ago regarding the late effects of certain infections. The early effects were more readily recognized clinically, while less importance was attached to the remote sequelæ except in so far as certain complications retarded individual recoveries. Little was thought then of the late effects of certain infections upon the mass-hygiene or sanitary condition of communities. For example, one decade ago but little importance was attached to the bacteriological condition of the child's throat subsequent to diphtheria, and the importance of the subject was not appreciated. It was hardly considered possible that many such children became chronic carriers of the infection for weeks or months subsequent to their attack. We now realize that a large percentage of the disease incidence arises in others from exposure to the mouth and throat secretions of such individuals. Nor in tuberculosis was it generally recognized that many individuals with chronic non-active types of the disease served as disseminators of the infection for periods of years, while themselves able to enjoy the blessings of apparent health.

Likewise, in typhoid fever, an ever present disease in our American municipalities, and in Asiatic cholera, the remote effects of these infections have only recently been appreciated. It is now generally recognized that about 4 per cent. of all typhoid cases become chronic carriers and disseminators of the infection for periods of months or years after their apparent recovery. It is entirely possible for the human body to harbor disease microörganisms of many other types for long periods of time without interfering with the apparent health of the individual. Sternberg discovered the micrococcus of pneumonia in healthy sputum, and every physician is familiar with the latent forms of syphilis and tuberculosis, which for long periods remain quiescent, and do not lead to destructive tissue changes. The various factors of immunity which protect the individual in such instances are not understood, but are generally covered by the explanation, so frequently given, but which serves merely to cloak our ignorance, the resistance of the individual and the varying virulence of different strains of bacteria.

In considering typhoid fever we have to deal with the following two modes of dissemination:

1. The spread of the disease by means of water, milk, and food products contaminated directly or indirectly by some active typhoid infection, but, as a rule, *remote* from the source of infection.

2. By contamination of water, milk, or food products *directly;* that is to say, by typhoid bacillus carriers who are in some way connected with the handling of food supplies.

We recognize two types of chronic typhoid carriers—"contact, idiopathic or primary carriers," who have never had the disease to their knowledge, and "secondary carriers," who have at some time previously had the disease.

PRIMARY, IDIOPATHIC, OR CONTACT CARRIERS. Typhoid carriers who become such through association with the disease, and such instances are not uncommon among nurses and orderlies, but who themselves have not had typhoid to their knowledge, are called "contact carriers." Since every typhoid patient requires the constant care of at least two or three individuals, it may readily be surmised that a large percentage of such attendants become "contact carriers." In many instances such individuals continue to excrete typhoid bacilli in the stools and urine for periods of years. Their own tolerance to the infection is probably due either to natural immunity or to a partial immunity from an earlier unrecognized mild typhoid infection.

Houston,[2] in 1899, reported the first case of persistent typhoid bacilluria in a contact carrier, who for three years had shown symptoms of chronic cystitis, while Drigalski and Conradi,[3] in 1902, found typhoid bacilli in the stools of 4 persons who had had no previous typhoid fever symptoms, but who had been in contact with typhoid fever. Park[4] has estimated "that probably one in every 500 adults who has never knowingly had typhoid fever is a typhoid-bacillus carrier." At first glance such figures are appalling, but with our recent knowledge of so-called "short duration typhoid" and of other factors, such as individual resistance and the varying virulence of different strains of bacteria, we can appreciate how many individuals at some time during their lives suffer an active typhoid without themselves being aware of it. In fact, it is entirely possible that many so-called summer diarrheas are in reality mild typhoid infections, which are not so diagnosticated. Such individuals receive a partial immunity, partial in the sense that, while themselves protected against the damaging influence of the infection and experiencing little subsequent disturbance, they are able to harbor, excrete, and perpetuate the bacilli in large numbers. Bacilli from such individuals when engrafted upon non-immune soil are able to produce all the clinical evidences of a severe attack.

SECONDARY CARRIERS. As above mentioned about 4 per cent. of all individuals who have, to their knowledge, had typhoid become chronic carriers of the disease for periods of months or years. They have secured from the previous attack only a partial immunity in the sense that while certain protective immune bodies, such as the opsonins and stimulins, are augmented by the attack, others, such as the bactericidins and bacteriolysins, for some unknown reason, are not augmented to the point of destruction of the bacilli. In other words, the factors which protect them individually from further damage do not lead to complete destruction of the infection, that is, their immunity is phagocytic, not bactericidal or bacteriolytic. Experience has shown that many typhoid bacillus carriers experience comparatively little discomfort. The attention of the physician is secured either because of their probable connection with endemic outbreaks or from individual symptoms, vague in character which lead to an investigation.

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[2] Brit. Med. Jour., January 14, 1899; ibid, October 9, 1909.

[3] Quoted by Park, Jour. Amer. Med. Assoc., September 19, 1908.

[4] Jour. Amer. Med. Assoc., September 19, 1908, p. 981.

Fütterer[5] in 1888 first called attention to the presence of typhoid bacilli in the gall-bladder, while Gilbert and Giroche, in 1890, are generally credited with the first description of typhoid bacillus cholecystitis. In 1893 Chiari[6] was able to collect 13 instances of typhoid bacillus cholecystitis and, in 1898, Cushing[7] tabulated 6 cases. The articles by these investigators and the paper by Ehret and Stolz[8] in 1900, in which 32 cases were described, had much to do with the recognition of the importance of the subject. Much discussion has arisen concerning the way in which typhoid bacilli reach the gall-bladder. The recent work of Chiari,[9] Hirch,[10] and Forster[11] and of Foster and Kazsen, would seem to convince most of us that the infection reaches the gall-bladder, in most instances, from the liver secretions or by way of the blood stream. Such mode of transmission undoubtedly occurs in contact carriers who have had a typhoid bacteriemia without the clinical evidences of enteritis, for Busse[12] has shown that the presence of typhoid bacilli in the blood stream do not necessarily give rise to enteritis or, for that matter, to agglutinating powers toward the bacilli. On the other hand, there is considerable evidence to show that in acute typhoid enteritis the bacilli may reach the gall-bladder from the duodenum. That typhoid ulceration may occur in the duodenum and stomach cannot be doubted. Such an instance of perforation of a typhoid ulcer in the duodenum occurred in the practice of my colleague, Dr. L. C. Grosh, in 1907.

After an attack of typhoid the infection may remain latent in the gall-bladder or in the alimentary or urinary tract for periods of years. In fact, with an earlier typhoid history, it is improbable that an acute exacerbation of latent cholecystitis is anything else but the result of the earlier infection. Levy and Kayser[13] found typhoid bacilli at autopsy in the gall-bladder of a patient who had no symptoms of cholecystitis, but who had had typhoid fever three years previously. Grimme[14] has reported the case of an insane patient who was isolated because of the presence of typhoid bacilli in the stools, and from whom numerous gallstones were removed at operation. After an interval following the operation, typhoid bacilli were no longer found in the stools. Hammond[15] found typhoid bacilli in the gall-bladder one year after an attack of typhoid. Hamilton,[16] who investigated 24 individuals with history of gall-bladder disease, found that 2 were typhoid bacillus carriers and 5 paratyphoid carriers. Fromme[17] has reported 4 cholecystectomies in 4 women typhoid carriers in all of whom elimination of bacilli ceased following the operation. Billings[18] has described the conditions present in a contact carrier who gave a positive Widal reaction and from whose stools a pure culture of typhoid bacilli was obtained. Upon operation for cholecystitis, calculi and typhoid bacilli were found.

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[5] Münch. med. Woch., 1888, p. 315.

[6] Präger med. Woch., 1893, p. 1.

[7] Johns Hopkins Hosp. Bull., 1898, ix, 91.

[8] Mitteilungen a. d. Grenzgebieten d. Med. u Chir., 1900, p. 389.

[9] Verhand. d. Deutsch. patholog. Gesellschaft, 1907, xi, 143.

[10] Ibid., xi, 150.

[11] Verhand. d. Deutsch. patholog. Gesellschaft, 1907, xi, 160.

[12] Münch. med. Woch., 1908, lv, No. 21.

[13] Ibid., 1907, liii, No. 50.

Holmes'[19] recent article on the subject also shows that cholecystitis due to typhoid bacillus can occur, as in one of Cushing's[20] patients, without the history of an earlier attack of typhoid. Such patients must be considered contact carriers. In Holmes' patient the bacilli disappeared from the bile and stools in eight weeks following cholecystostomy. Holmes has very properly brought up the point that the mere presence of typhoid bacilli in the gall-bladder is not of itself sufficient to provoke the clinical symptoms of cholecystitis. It is only when the cystic or common duct is obstructed, during perhaps an otherwise latent period of the gall-bladder infection, that symptoms manifest themselves which we recognize as the clinical evidences of cholecystitis.

In most chronic carriers the bacilli are eliminated with the feces; in a smaller number the urine contains the bacilli. Forster believes the gall-bladder to be the site of continued reproduction of the bacilli which are intermittently ejected into the intestine. The Ledinghams uphold this view, since investigations have shown that the bacilli may disappear during the convalescent period of the disease only to reappear several months later, following perhaps, the administration of some cholagogue cathartic as suggested by Reitter.[21]

The History of Typhoid Bacillus Carriers. Experience has shown that, in most cases, the discovery of typhoid carriers has arisen from hitherto unexplainable endemic outbreaks among the inmates of asylums, or among families who have happened to employ the same cook, or obtained milk from a dairy previously under suspicion. A large number of typhoid carriers, in the sense that they have been the cause of such endemics, have been women who were, in some way, connected with the handling of food products. On the other hand, many carriers exist who, rather from good luck than good management, do not happen to infect others. Any individual who has been in contact with the disease, or who has himself suffered an earlier infection, may be a carrier in the sense that pure cultures of the bacilli may be obtainable from the stools or urine. Probably not more than one-half of such carriers actually infect others, although no definite data is available on this point, since the matter of cleanliness in personal habits varies, as a rule, with the social status of the invividual.

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[14] Münch. med. Woch., 1908, lv, No. 1.

[15] Jour. Amer. Med. Assoc., January 2, 1909.

[16] Ibid., February 26, 1910.

[17] Deutsch. Zeitschr. f. Chirurg., 1910, cvii, No. 6.

[18] Trans. Amer. Med. Assoc., June 27, 1911; abst. Medical Record, New York, 1911, lxxx, No. 1.

[19] New York Med. Jour., August 12, 1911.

[20] Loc. cit.

[21] Wien. med. Woch., 1908, p. 519.

Until the recent communication of Young, mentioned below, the largest number of recorded cases originating from one source was reported by Lumsden and Woodward,[22] who found a typhoid carrier in a dairy responsible for 54 cases of the disease among the dairy customers. This carrier had had her attack eight years before. The time element since the original typhoid attack has been found to vary greatly. Gregg[23] has reported the case of a boarding-house mistress, who served as the source of infection in 7 cases of typhoid, fifty-two years after her own recovery. No bacilli were found in the blood or urine, but pure cultures were obtained from the feces. Jundell[24] has described the conditions present in a family, the grandmother of whom was known to be a typhoid carrier for fifty-four years, and during this interval 22 members of the family were attacked. This carrier was eighty-three years old, and her feces were found to contain typhoid bacilli.

Scheller[25] has described an endemic in Prussia in which during a period of fourteen years, 32 cases of typhoid were traceable to a woman employed in a dairy whose attack had occurred seventeen years previously. Typhoid bacilli were found in her stools in pure culture. Out of 40 people who drank the milk of this dairy, 18 were found to be carriers of the infection, and yet only 5 of the 18 had ever had typhoid. In other words, 13 were contact carriers. Soper's[26] painstaking investigation of so-called "Typhoid Mary," a contact carrier, showed that in ten years, 26 cases of typhoid could be traced to her as cook in households where the disease appeared. Typhoid bacilli were found in the stools, but none in the urine.

Young,[27] Commissioner of Health, of Chicago, in a personal communication, has recently described the conditions which resulted from the employment of a young woman in one of the suburban dairies. In June and July, 1911, three years after her apparent recovery from typhoid, 56 cases of typhoid were found in families receiving their milk supply from this dairy. To July 21, 1911, 4 deaths had occurred among this number. Negative bacteriological results were obtained in the study of the bowel and bladder discharges of all individuals connected with the dairy, except in the case of the young woman above mentioned. Part of her duty was to wash the milk cans. Nieter and Liefmann[28] found 7 carriers among 250 inmates of an insane pavillion where typhoid was endemic. At the autopsy of 1 patient who succumbed to chronic dysentery, typhoid bacilli were found in the intestines and in pure culture in the gall-bladder. The serum of this patient would agglutinate the typhoid bacillus in dilution 1 to 50, and the Flexner dysentery bacillus in dilution 1 to 100. After isolation of the carriers, the endemic ceased. Busse[29] was able to locate typhoid bacilli from the blood of 4 carriers who died of intercurrent diseases. Hutchinson[30] has reported the career of a lodging-house keeper whose attack occurred fifteen years earlier and who was responsible for 7 cases of typhoid. Similar investigations have been made by the Ledinghams,[31] by Connell,[32] Huggenberg,[33] Southward,[34] Dehler,[35] Nieter,[36] Branthwaite,[37] and by Madsen.[38]

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[22] Jour. Amer. Med. Assoc., March 6, 1909, p. 749

[23] Boston Med. and Surg. Jour., July 16, 1908.

[24] Hygiea Festband, 1908; abst. edit. Jour. Amer. Med. Assoc., January 30, 1909, p. 388.

[25] Centralbl. f. Bakteriol, 1908, No. 5, p. 385; abst. edit. Jour. Amer. Med. Assoc., June 13, 1908, p. 1986.

[26] Jour. Amer. Med. Assoc., June 15, 1907.

[27] Personal Communication, July, 1911.

Diagnosis. It will be obviously impossible to keep any large number of typhoid convalescents under bacteriological surveillance. As mentioned in an earlier article[39] popular education may be of service in dealing with the problem. This education can best be carried on by physicians who should bear in mind the possible connection between an earlier attack of typhoid, or the possibility of infection by contact, and symptoms, sometimes slight, referable to the gall tract, to a slight urinary cystitis, or bacteriuria, to chronic colitis, to mild dysentery, or to attacks of so-called intestinal indigestion. The source of endemic outbreaks should be investigated whenever possible, and the possibility of direct infection of food products by a typhoid bacillus carrier should be borne in mind. Surgeons should have cultures taken in all operations involving the gall-bladder, and physicians at the time of their examinations should search for post-typhoidal sequelæ in those who have had the disease, and for contact carriers through association, in those who deny previous infection. Such sequelæ are especially apt to be found among patients presenting evidences of gall-bladder disease, the symptoms of which are frequently referred to the stomach. In fact, all patients showing evidences of gall-gladder disease, whether months or years after a history of typhoid, should be considered as possible chronic typhoid carriers until proved otherwise.

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[28] Münch. med. Woch., 1906, liii, No. 33.

[29] Loc. cit.

[30] Brit. Med. Jour., March 26, 1910.

[31] Ibid., January 4, 1908; ibid., October 17, 1908.

[32] Amer. Jour. Med. Sci., 1909, cxxxvii, 637.

[33] Correspbl. f. Schweiz. Aerzte, 1908, xix, 635.

[34] Archives of Pediatrics, March, 1907.

[35] Münch. med. Woch., 1907, liv, No. 16; ibid., 1907, liv, No. 43.

[36] Ibid., 1907, liv, No. 33.

[37] British Government Report, abst. Jour. Amer. Med. Assoc., April 4, 1908.

[38] Hospitalstidende, Copenhagen, 1910, liii, No. 14; abst. Jour. Amer. Med. Assoc., June 25, 1910.

[39] Jour. Amer. Med. Assoc., 1910, lv, 1708.

Cultures should be taken in all post-typhoid cases whose urine contains bacteria. Likewise cultures should be taken from the stools in all cases showing symptoms of chronic dysentery or colitis. The colon bacillus will practically always be obtained from the stools, and may be confusing, but the typhoid bacillus, if present, can be isolated from the plates with comparatively little difficulty. The diagnosis will depend upon the isolation of a bacillus which gives culturally the main characteristics of the bacterium of Eberth. That is to say, a short motile rod which grows rapidly in bouillon or on agar slant, which does not liquify gelatin nor produce indol or coagulate milk, nor produce gas or acid in glucose litmus gelatin, and produces an invisible growth on potato.

It is the personal conviction of the writer that these main characteristics are not given by all strains of typhoid bacilli, for in 1 of the 3 cases studied during the past two years the cultural findings were confusing. In one (C. R. D.) who had never had typhoid and who was regarded as a contact carrier, the pure cultures obtained from the urine throughout a long period of treatment, would at times correspond to the main cultural characteristics mentioned, while again slight amounts of gas and acid would be produced in the media. In this connection it may be worth mentioning that no symptoms of cystitis or pyelitis were present in this patient. He was referred for examination because of the constant presence of enormous numbers of bacteria in the urine. During many months of treatment no pus cells were found, a condition seldom, if ever seen, when the colon bacillus is the causative organism.

In other words, the colon bacillus when present in the bladder produces an inflammatory reaction which is recognized as a true cystitis, while the typhoid bacillus when present in the urinary tract, in typhoid carriers is eliminated in suspension in the urine, a condition recognized as bacteriuria. Still further proof, in support of the view that this patient was a typhoid carrier, is afforded by the fact that he failed to react to the treatment ordinarily successful in dealing with colon bacillus infections of the urinary tract; that is, the use of hexamethylenamine and sodium benzoate internally and the use of a colon vaccine. It may be mentioned also that his blood serum would agglutinate, in dilution 1 to 50, a stock typhoid culture. He was not benefited by inoculations of an autogenous vaccine, and gave up treatment before inoculations of a stock typhoid vaccine were begun.

The agglutinating power of the blood serum as well as the phagocytic power is found to be increased in typhoid bacillus carriers, as a rule, while the bactericidal and bacteriolytic powers are below normal. This is in accord with the previously expressed opinion that such individuals are but partially immune, since it has been shown[40] [41] that normally during convalescence from the disease, the bactericidal and bacteriolytic properties of the serum are increased to the point of destruction of the bacilli. These facts are of value in the bacteriologic study of a suspected individual and may be of service from a diagnostic standpoint. Alice Hamilton[42] has also shown that carriers have a persistently high opsonic index and relatively high agglutinative power; that is, their immunity is phagocytic, not bacteriolytic, or bactericidal.

Report of a Typhoid Carrier and Vaccine Treatment. R. F. (record 565), aged twenty-eight years, from Deerfield, Michigan, was referred by Dr. R. M. Eccles, January 17, 1911, as a possible typhoid carrier, although no cases of the disease were traceable to him. In 1904 this patient had typhoid fever for six weeks with apparent recovery for one year, after which time he began to complain of pain in the region of the umbilicus which continued until 1909. The subsequent history was supplied by Dr. Geo. Sellards, of Deerfield. In 1909, diarrhea began; the bowel discharges numbered five or six daily with some mucus, and in December, 1909, three severe hemorrhages from the bowel occurred, with temperature as high as 104°. The last hemorrhage measured over one quart. The diarrhea continued for one year thereafter. The discharges were occasionally streaked with blood and mucus but gradually subsided under medication. The weight before the attack of typhoid seven years ago was 151. At the time of examination it was 134. He complained of frequent headaches. The examination revealed nothing of importance on the part of the heart, lungs, nervous system, and abdomen, except some tenderness on pressure was present below the umbilicus. One loose stool occurred every morning, with poor control of the sphincter ani. Examination of the fresh stool for Amebæ dysenteriæ, as well as stains of the mucus present for tubercle bacilli were negative. The von Pirquet skin reaction was negative. The leukocytes numbered 7200, and the urine was negative to albumin, pus, and sugar. There had never been any urinary symptoms. The Adler occult blood test was positive in the stools. Cultures taken from the stools showed the presence of the colon bacillus and a short, plump, actively motile rod, which in pure culture, did not produce acid in litmus gelatin, or gas in stab culture on glucose agar, nor was indol produced after ten days growth in bouillon. Milk was not coagulated. The agar slant showed a growth which resembled that of the typhoid bacillus, while the growth was invisible on potato. Gelatin was not liquefied in six weeks. Cultures from the urine showed a few isolated colonies of Staphylococcus albus (probably contamination). Subsequent cultural studies of the fresh stool gave the same findings. His blood serum would agglutinate a stock typhoid culture in dilution 1 to 20, while the autogenous strain would agglutinate in dilution 1 to 10.

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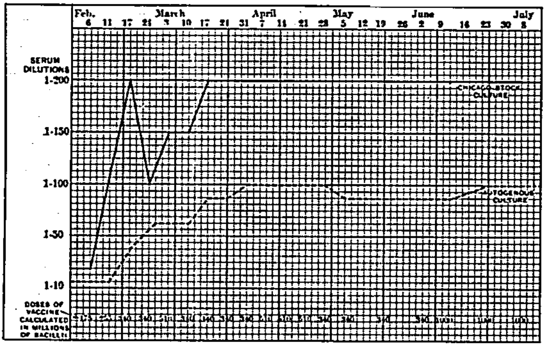
[40] Proc. Royal Soc., 1903, and Jour. Royal Army Med. Corps, 1907 and 1908.

[41] Jour. Amer. Med. Assoc., 1909, liii, 1253.

[42] Loc. cit.



Mr. R. F., aged twenty-eight years. Chart shows the increase of the agglutinating power of his serum for the bacillus of Eberth under injections of autogenous typhoid vaccine.

A vaccine was prepared from the typhoid organisms present in the stools. He received his first inoculation on February 6, 1911. Between that date and May 5, 1911, fourteen inoculations were given at weekly intervals, the doses varying between 110,000,000 and 510,000,000. Negative cultures were obtained in May, 1911. Since that time he has received five additional inoculations, three of which have contained 1,000,000,000. The agglutinating power of the patient's serum was increased ten fold during the course of treatment. Coincident with the vaccine treatment and the increase in the agglutinating powers of his serum (see Chart) his general condition improved. The tendency to loose stools with poor sphincter control disappeared and the bowel discharges became normal in appearance and gave negative cultures. He was discharged July 8, 1911.

THE MEDICINAL TREATMENT AND CONTROL OF TYPHOID CARRIERS. The problem is a perplexing one, since the installation of adequate filtration plants and careful dairy supervision will not, for years to come, rid most American municipalities of typhoid. In most localities the disease has been endemic for years with a consequent large number of unrecognized carriers. Such individuals if employed as cooks, waiters, and waitresses, dairymen and dairymaids, workers in confectionery, hucksters, workers in artificial ice plants, or in any capacity which requires the handling of food products may continue to serve throughout long periods of time as a menace to the public health.

Since the treatment of such individuals has not been uniformly successful, the question immediately arises as to what steps local and state boards of health should employ to protect the public. For example, in the recent Chicago outbreak, 56 cases of typhoid with 4 deaths have resulted from the uncleanly personal habits of a young woman employed to wash the milk cans in the dairy. What other attitude could or should be taken by the Municipal Department of Health except to employ its police powers toward the isolation of such an individual for treatment. Although innocent of harm she has been directly responsible for 4 deaths to date, to say nothing of the invalidism and expense incurred by over 50 others. There is much to be said regarding personal liberty, but since the law gives protection to property, personal and real, it is only fair to assume that such protection should be given to the health of individuals who are well; an asset far greater than the asset of property. Progress in every walk of life depends upon those who are in good health, but strange to say, the wheels of progress turn slowly, and stranger still, our law-making bodies can be induced to attach relatively little importance to prophylactic medicine. However, the danger to the public health will be greatly minimized, if when typhoid bacillus carriers are discovered control could be exercised over them by the local boards of health, with the idea of securing proper treatment, and restricting them to employment which do not have to do with the handling of food supplies.

The treatment of the chronic typhoid carrier by so-called intestinal and urinary antiseptics, such as sodium phenolsulphonate, sodium salicylate, salol or hexamethylenamine, and sodium benzoate, has been of little avail. During convalescence from the disease when the natural immune bodies, such as the bacteriolysins and bactericidins, are augmented, such antiseptics are undoubtedly of value and should be employed in every case as an useful adjunct. Graham, Overlander, and Dailey[43] found that 23 per cent. of 65 typhoid patients showed typhoid bacilli in stools and urine for ten

[43] Boston Med. and Surg. Jour., January 14, 1909.

days before their discharge. An important lesson should be learned from these facts, since it is entirely probable that if the convalescence of typhoid cases was studied and treated with as much care as the active stages of the disease, the number of chronic carriers would be greatly lessened. During convalescence from the acute attack, when conditions are most favorable because of the natural augmentation of immune bodies, help may be secured by medicinal means; while in chronic carriers the bactericidal and bacteriolytic properties of the blood serum are decreased and such medicinal assistance as may be offered, is of little avail.

VACCINE TREATMENT. The number of typhoid carriers successfully treated by vaccines has not been large. Irwin and Houston[44] were able to cause disappearance of the infection in a patient who for seven years had been a carrier. She received during two months, five injections varying from 50,000,000 to 500,000,000. Meader[45] has also successfully treated a patient and found that the best therapeutic dose varied between 75,000,000 and 400,000,000. He has shown that the disappearance of the infection coincided with the increase in the bactericidal power of the blood serum of the patient. In my report[46] of a typhoid carrier treated during 1910, it was shown that the agglutinative, bacteriolytic and bactericidal properties of the blood serum were augmented during the course of six injections which varied from 100,000,000 to 400,000,000. The increase in the immune bodies of the serum was followed by the disappearance of the infection. Brem and Watson[47] have recently reported the case of a child, aged four and one-half years, whose urine was found to contain the bacilli a few months after convalescence. She subsequently received nine injections of autogenous vaccine in about two months, when the urine was found to contain no bacilli. The agglutinative powers of the patient's serum increased markedly to 1 in 1000 during the course of inoculations.

In fact, it may be said, that the treatment of carriers by inoculations of vaccine offers more hope for cure than any other known method. It probably is better to use a vaccine prepared from the patient's own organisms, although no definite proof exists that such is the case. The work of Leishman and his associates seems to prove that as efficient an immunity is conferred by a stock vaccine as by an autogenous vaccine, and that a single nonvirulent strain is capable of producing as efficient an immunizing response as vaccine prepared from single or multiple virulent strains. The method of dosage found to be most satisfactory has been to increase gradually from 100,000,000 to 1,000,000,000 depending upon the evidences of immunizing response as obtained by study of the agglutinating, bactericidal, and bacteriolytic power of the blood serum.

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[44] Lancet, London, January 30, 1909.

[45] Johns Hopkins Hosp. Bull., September, 1910.

[46] Loc. cit.

[47] Trans. Amer. Med. Assoc., June 27, 1911; abst. Medical Record, New York, 1911, lxxx, No. 1.

The vaccine should be prepared as follows: Pure cultures of the organisms are grown on agar for twenty-four hours, when the growth is washed off with sterile salt solution. The resulting suspension is thoroughly shaken to break up the clumps and a small quantity drawn into an ordinary red blood pipette, diluting 1 to 200, with salt solution for purpose of standardization. A small drop of this dilution is then placed upon the *blood-platelet* counter of Zeiss. The *blood-platelet* counter differs from the ordinary red blood cell counter in that with the former, the depth of the chamber is only $\frac{1}{50}$ mm. Twenty-five small fields are counted under the microscope, using dim direct illumination or indirect illumination by the dark-field condensor, and the sum divided by twenty-five to obtain the unit value. The unit value is then multiplied by the dilution, by the depth of the chamber, by 400, the number of small squares in a millimeter, and, lastly, by 1000 to convert to cubic centimeters. For example, if fifty bacterial cells are counted in twenty-five small squares, the unit value per square would be two, multiplied by 200, by 50, by 400, and by 1000, or 8,000,000,000, the number of bacteria per c.c. of suspension. The suspension is then heated to 54° to 55° C. for one hour in a sealed test-tube, in a water bath and then diluted with sterile salt solution, containing 0.35 per cent. phenol, so that each cubic centimeter of the finished vaccine contains approximately 1,000,000,000. Care must be exercised not to overheat the vaccine, since overheating impairs its immunizing properties. The thermal death point of most strains of typhoid bacilli varies between 53° and 56° C. Cultures should be taken from the vaccine to insure sterility before using.

CONCLUSIONS. 1. Not only should individuals who are typhoid carriers receive appropriate vaccine treatment, but all individuals including physicians and nurses, whose duties require the attendance and care of typhoid cases should receive immunizing treatment. By this procedure many individuals otherwise destined to become chronic carriers might secure protection which would prevent in themselves the possibility of cholecystitis or other late manifestations, as well as avoid the possibility of becoming a source of danger to others.

2. Such treatment has been found efficient and productive of little discomfort. Nurses, orderlies, and physicians should be regularly immunized at intervals of two or three years, as should all others exposed to endemic or epidemic outbreaks.

3. When typhoid bacilli have produced clinical or bacteriological evidences of cholecystitis, it is doubtful whether vaccine treatment will of itself be of avail, surgical drainage of the viscus then being indicated. On the other hand, appropriate vaccine treatment in

individuals showing evidences of intestinal or urinary tract perpetuation of the bacilli, without cholecystitis, during convalescence from the disease or subsequently, should be given the opportunity of vaccine treatment. Not all will be cured but the bacilli will disappear in some instances.

4. Chronic carriers who have been unsuccessfully treated by vaccines should be under the control of the local board of health and furnished employment if necessary which would not require the handling of food products.

EPIDEMIC POLIOMYELITIS: A CLINICAL STUDY OF THE ACUTE STAGE.[1]

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It is an interesting fact, that although our knowledge of acute poliomyelitis dates back over seventy years, when the German surgeon, Jakob Heine[2] first described it under the name of "the spinal paralysis of children;" the true nature of the disease, its pathology, clinical manifestations, prognosis, infectiousness, contagiousness, have only lately been fully ascertained, and have, only in the last decade, become the subject of general knowledge. Furthermore, it is of interest to note that at the same time that Heine first made his observations in Germany, an American practitioner, Dr. Colmer, observed a small epidemic in Louisiana, and recorded it in the Amer. Jour. Med. Sci. (1843, v, 248). Little or nothing was added to the knowledge of the subject for the next thirty years. No epidemics were reported until 1881, when 18 cases occurred in Sweden. Subsequently, epidemics were reported from Norway, Sweden, Germany, the United States, and Canada, in the eighties and nineties. The most extended epidemics occurred in the above-named countries in the last five or six years.

A noteworthy contribution was made by Strümpell[3] in 1885, in his paper on Poliencephalitis Acuta in Children. Pathologically and clinically this disease was noted as identical with the well-known forms of spinal poliomyelitis. For the first comprehensive clinical studies we are indebted to the Danish physicians Medin[4] and Wickman.[5] Aside from his contributions to the pathology, Wickman was able to show conclusively, that the disease is contagious.

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[1] Read before the Metropolitan Medical Society, Academy of Medicine, October 24, 1911.

[2] Die spinale Kinderlaehmung, 1840 and 1860: a study of 150 cases.

[3] Jahrb. für Kinderheilk., 1885, Band xxii, N. F.

[4] X. Internationaler Congress, Berlin, 1890.

[5] Beiträge zur Kenntniss der Heine–Medinschen Krankheit, Berlin, 1907.